

Ionizing Radiation and Its Risks

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Penetrating ionizing radiation fairly uniformly puts all exposed molecules and cells at approximately equal risk for deleterious consequences. Thus, the original deposition of radiation energy (that is, the dose) is unaltered by metabolic characteristics of cells and tissue, unlike the situation for chemical agents. Intensely ionizing radiations, such as neutrons and alpha particles, are up to ten times more damaging than sparsely ionizing sources such as x-rays or gamma rays for equivalent doses. Furthermore, repair in cells and tissues can ameliorate the consequences of radiation doses delivered at lower rates by up to a factor of ten compared with comparable doses acutely delivered, especially for somatic (carcinogenic) and genetic effects from x- and gamma-irradiation exposure. Studies on irradiated laboratory animals or on people following occupational, medical or accidental exposures point to an average lifetime fatal cancer risk of about 1×10^{-4} per rem of dose (100 per 10^6 person-rem). Leukemia and lung, breast and thyroid cancer seem more likely than other types of cancer to be produced by radiation. Radiation exposures from natural sources (cosmic rays and terrestrial radioactivity) of about 0.1 rem per year yield a lifetime cancer risk about 0.1 percent of the normally occurring 20 percent risk of cancer death. An increase of about 1 percent per rem in fatal cancer risk, or 200 rem to double the "background" risk rate, is compared with an estimate of about 100 rem to double the genetic risk. Newer data suggest that the risks for low-level radiation are lower than risks estimated from data from high exposures and that the present 5 rem per year limit for workers is adequate.

In the 87 years since the discovery of radiation and radioactivity, considerable information has become available concerning the characterization and quantification of radiation and its interaction with matter. More is probably known about the consequences of exposure to ionizing radiation than any other environmental or occupational hazard. Paradoxically, the increase in knowledge about the consequences of radiation exposure has increased its ranking as an issue of serious concern to society.

This brief overview will attempt to highlight some of the basic radiobiologic concerns that apply to the assessment of population and occupational exposures. It includes some discussion of the ways in which radiation risk can be quantified and assessed, and some areas of future concern.

The Nature and Measurement of Ionizing Radiation

The unique electrical nature of ionizing radiation renders it particularly amenable to easy detection and quantification. Regardless of the source, the absorption of ionizing radiation by matter is accompanied by the creation of ion pairs—that is, the physical disruption of neutral atoms caused by the dislodging of the target atom's orbital electron. The dislodged electron and the residual electron-deficient atom constitute an ion pair, and the process is known as ionization. This process can occur when electromagnetic or particulate radiation is absorbed in any target.

The kinds of exposures of principal concern to society are primarily from x-irradiation, gamma-irradiation and beta particle irradiation. These three types of

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ABBREVIATIONS USED IN TEXT

LET=linear energy transfer
RBE=relative biologic effectiveness

ionizing radiation all emit low linear energy transfer (LET) radiations, meaning that they have in common a characteristically sparse path of ion pair generation. Thus as radiation penetrates matter, it creates ion pairs with relatively large distances between them. Frequently these ion pairs appear in clusters. By contrast, high-LET radiation is characterized by relatively short range, very intense, closely patched ion pairs.¹

The number of ion pairs absorbed within a volume of tissue constitutes the absorbed radiation dose and we quantify this absorption of electrical energy in terms of rads (radiation-absorbed dose). The absorption of 100 ergs of ionizing radiation in 1 gram of matter has a value equal to 1 rad. A new System Internationale (SI) nomenclature for radiation units has been adopted, and the term rad is now being replaced by an absorbed dose unit called the gray (Gy). One rad equals 0.01 Gy (specifically, 1 Gy=1 joule/kilogram). The rad is a physical radiation absorbed dose unit which, when multiplied by a relative biologic effectiveness factor (RBE), permits one to account for the different biological effectiveness of radiations of different LET. The product of RBE times rads equals rem (rad equivalent in man or mammal). The SI replacement for the rem is the sievert (Sv), and 1 rem equals 0.01 Sv.

For purposes of continuity and convenience, rads or rems will be used in this discussion. Since the radiation discussed to the greatest extent is entirely of low-LET quality with an RBE value of unity, we can, in this context, use rads and rems interchangeably when considering x-, gamma- and beta-irradiation.¹

Assessing radiation hazards requires knowledge not only of the essential characteristics of the quality of radiation absorbed by target tissues but also its distribution in space and time. As with exposures to many other substances, it has been observed in radiation research that as the doses absorbed by individuals increase, the magnitude and consequences of the exposure also increase. The rate with which a given dose of radiation is absorbed frequently plays a significant role in the manifestation of radiation effects.²

Some perspective on the magnitude of radiation doses might best begin with consideration of the annual background radiation dose to which all of us are exposed. This background radiation exposure is the result of cosmic radiation, of natural radioactivity from the earth's mantle beneath us and of the small amount of natural radioactivity that is present in all living tissues. The natural background radiation dose rate in the United States generally averages approximately 0.1 rem annually.³ It can increase by about a factor of two in certain parts of the country where underlying terrestrial radioactivity might be slightly higher due to different geologic formations, and where, at high altitudes, the thinner atmosphere permits a slightly higher flux of

cosmic radiation. For example, the annual background rate in California is approximately 0.1 rem per year, but in Denver, Colorado, it is about 0.2 rem per year. There are parts of France where the natural background rate is 0.5 rem per year, and in certain locales in Brazil and India it may be as high as 1 rem per year. It is evident that on this planet there is no such thing as a radiation-free environment.

It is realistic to assume over the course of our lifetimes we each accumulate radiation doses from background exposures of between 5 and 10 rem at exceedingly low rates. Although present estimates vary somewhat, if in addition one were to average all of the radiation exposures from the healing arts, consumer products and all other man-made sources, our average absorbed dose rate would double. Therefore, a typical American would receive approximately 0.2 rem per year, half of it natural and half of it man-made.⁴

There is no direct evidence to support any medical consequences from exposures to background radiation. Only when the radiation dose is greatly increased are the consequences measurable.^{1,4}

Basic Interaction of Radiation with Biologic Targets

Ionizing radiation produces its effects by either directly ionizing and disrupting a molecule of critical importance to the cell (usually in the cell nucleus) or through the production of highly reactive free radicals (usually of water). These direct and indirect effects produce the initial lesion which may be amplified to produce radiation effects. Nonionizing forms of radiation can cause resonance or excitation of impacted atoms, but only ionizing radiation produces ion pairs.⁵ Ionization has serious consequences in certain cellular structures. Many theories and much research address the sequence of amplifying steps from the initial atomic reaction following absorption of ionizing radiation to the manifestation of a clinical entity such as the initiation of a cancer or the creation of a genetic defect. The serious occupational health problems resulting from ionizing radiation stem from the killing of cells consequent to large doses of radiation, or from nonrepaired injuries in the form of molecular lesions in cells, primarily within the nucleus, that survive the radiation.

Cell and Tissue Radiosensitivity

The results of low LET radiation exposures of single cell preparations in vitro show that cell survival and reproductive capacity can be diminished by relatively high radiation doses.⁵ Laboratory studies of human, animal and plant cells have shown that exceedingly small radiation doses given at very low dose rates do not necessarily produce an effect that is linearly proportional to the radiation dose.⁶ In the lower dose domain, the data are consistent with the assumption that some of the initiating molecular events after the absorption of radiation doses are repaired, or "recovered," to some extent. To the extent that any repair occurs within these cells, and that this repair efficiency is dose related, the dose response will likely not be strictly proportional over the full range of tested radi-

ation exposures.⁷ As an example, when the radiation dose is quite low, as from the slow decay of a deposited long-lived radioactive element, a long time may be required and a substantial dose may be necessary before low-LET radiation-induced cancer can be found. At higher doses, radiation-induced cancer is likely to appear sooner.⁸

Different cells and tissues appear not to be quantitatively similar in their response to radiation exposure. About 80 years ago, Bergonie and Tribondeau observed that the sensitivity of cells to radiation seemed to be greatest in those cells that were most primitive, that

divided most frequently and had the shortest interval between divisions. Cells of reproductive organs, cells lining the intestinal tract and the primordial cells of the bone marrow have these characteristics. Muscle and neuron cells are uniquely radioresistant, highly complex and nonmitotic.⁹ As an aside, it is an interesting observation that cells in tissues that divide most rapidly do not necessarily correlate with potential radiation-induced cancer risk.

Acute, Latent and Genetic Radiation Effects

If uniform radiation is absorbed by the body, a spectrum of effects is seen, each related in time and extent to the magnitude of radiation dose. Figure 1 illustrates a temporal sequence of changes in blood sampled from a person receiving a nearly lethal dose of radiation.⁹ Note that the different cell types respond differently to the radiation exposure and that there are cell populations, in this case the bone marrow, that show some recovery towards the end of the sequence.

Identification of acute radiation effects can be summarized as shown in Table 1. It should be noted that if a hypothetical population were to receive a large exposure to radiation, and the population was sufficiently small, it is possible for medical intervention to ameliorate the severe consequences. It is a radiobiologic fact of life, however, that radiation-induced lesions and their consequences at high doses are to a very large extent nonreversible.¹⁰ Large total-body doses of radiation impact most severely on the regenerative cells of the intestinal epithelium, on the regenerative cells of the bone marrow and on the microvascular system's endothelial cells. As a consequence, soon after irradiation a number of effects evolve in which phagocytic function is impaired, in which electrolyte containment within the body is compromised, and in which gastrointestinal integrity due to massive hemorrhage and cell depletion permits bacterial entry into the body from the gastrointestinal contents. The resulting loss of hematopoietic defenses, vascular integrity and electrolyte imbalance at radiation doses in excess of 300 to 400 rads is likely to be considered lethal.¹⁰ Therapy in the form of antibiotic attack on bacterial infection, cell transfusion and even in some instances bone marrow transplantation has some moderate effectiveness. However, the micro-anatomic disruption of the body's defenses is not easily repaired by external sources and at best therapy constitutes a holding action until the surviving cells in the body can repair the initial damage. Furthermore, despite a massive research investment over the past three decades, there have been no successful preventive medications developed that could act as a competing target for the effects of ionizing radiation, and thereby minimize its impact.⁵

Early in the evolution of radiobiologic research, effects of radiation at doses lower than those needed for acute, immediate effects were frequently quantified in terms of life-shortening or acceleration of aging.¹ We now know that this to a very large extent appears to be another means of quantifying an increase in the acceleration of cancer risk statistics in populations exposed

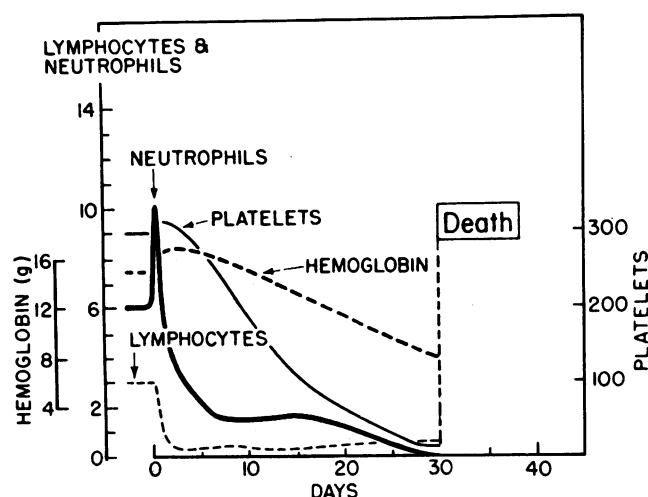


Figure 1.—Typical hematological response of human beings to total-body radiation dose of 450 rads. Lymphocyte, neutrophil and platelet values should be multiplied by 1,000. Hemoglobin values are in grams per dl. (Reproduced with permission from Andrews GA: Radiation accidents and their management. Radiat Res 1967; 7(Suppl):390.)

TABLE 1.—Acute Effects of Whole Body Irradiation

REM	
5-20 Possible late effect; possible chromosomal aberrations
20-100	... Temporary reduction in leukocytes
100-200	... "Mild radiation sickness"; vomiting/diarrhea/tiredness in a few hours Reduction in infection resistance Possible bone growth retardation in children
200-300	... "Serious radiation sickness"; bone marrow syndrome; hemorrhage; LD _{10-35/30} *
300-400	... "Serious radiation sickness"; marrow/intestine destruction; LD _{50-70/30}
400-1,000	.. Acute illness, early deaths LD _{50-75/30}
1,000-5,000	.. Acute illness, early deaths in days; intestinal syndrome LD _{100/10}
5,000+ Acute illness; death, early deaths in hours to days; central nervous syndrome LD _{100/2}
Also	
50+ in men yields temporary sterility (100 rem+ = 1 year duration)
300+ in women yields permanent sterility

REM=rad equivalent in man or mammal; LD=lethal dose.

*Lethal dose to percentage of the population in number of days (for example, LD_{10-35/30}=lethal dose in 10 percent to 35 percent of the population in 30 days).

to small to intermediate levels of radiation, and not to some unique change in physiology.

If radiation is protracted, its consequences are usually less severe than when the same radiation dose is delivered acutely.⁷ For example, in a study on beagle dogs carried out at our laboratory some three decades ago, it was noted that when a dose of 300 rads of radiation was absorbed by dogs, the animals with the greatest interval between exposure fractions and the smallest radiation dose per fraction had decreased life-shortening or a lower cancer rate than animals receiving the same total dose in shorter intervals, or larger fractions.¹¹ The magnitude of this dose-rate amelioration factor was approximately a factor of 3 at its greatest (that is, a given dose via protracted radiation was a third as carcinogenic as the same dose given acutely). Another study now nearing completion in our laboratory on the effects of strontium 90, a low-LET beta-particle-emitting radionuclide that concentrates in the skeleton, shows that bone cancer is induced only when the radiation dose rates exceeded approximately 1 rad (or rem) per day, and then only after several years of exposure (that is, several hundred rems of dose).¹² A 1,500-fold range of doses was tested from about 7 millirads to 15,000 millirads per day, showing at the higher levels a full range of myeloproliferative disorders of marrow, as well as cancer of bone. The dose-response relationship does not appear to be a straight line, but is curvilinear. That

is to say the curve is sigmoid or S-shaped, being very shallow at low doses and rising in a nonlinear fashion to a maximum level of effect at about 5 or more rads per day. Cumulative radiation doses associated with the radiation-induced cancers are above about 1,000 rads. The point to be made is that while cancer incidence is expected, it took rather large doses at low rates to be induced. Again, this appears to confirm that low dose rates are less efficient than high dose rates for the same total dose regarding the risk for cancer induction.

Recently the National Council on Radiation Protection and Measurements has cited tumorigenesis data on some 12 different studies in laboratory animals covering doses ranging up to 200 or 300 rem.⁷ These all show that low-dose rate radiation is about two to ten times less carcinogenic for the same doses compared with radiation delivered at high rates. It is important to reiterate at this point that in the intermediate- and low-dose range, the principal, if not sole, radiation effect in the exposed population is the somatic risk increase for cancer. An increase in this risk in an exposed population can cause shortening of the lifespan.

Today the challenge to scientists, especially to epidemiologists and biophysicists, is the methodology involved in the computation of accurate risk estimates. Of considerable complexity, this health risk estimation is always done in a manner complicated by the way in which the normal cancer statistics of the population at risk are to be handled. Approximately 1,700 people per million each year die of cancer from all causes.¹⁰ It is known that this disease is predominately one of old age, as shown in Figure 2, in which most types of cancer have an almost exponential increase with age.⁴ It is on this natural history of cancer incidence and mortality

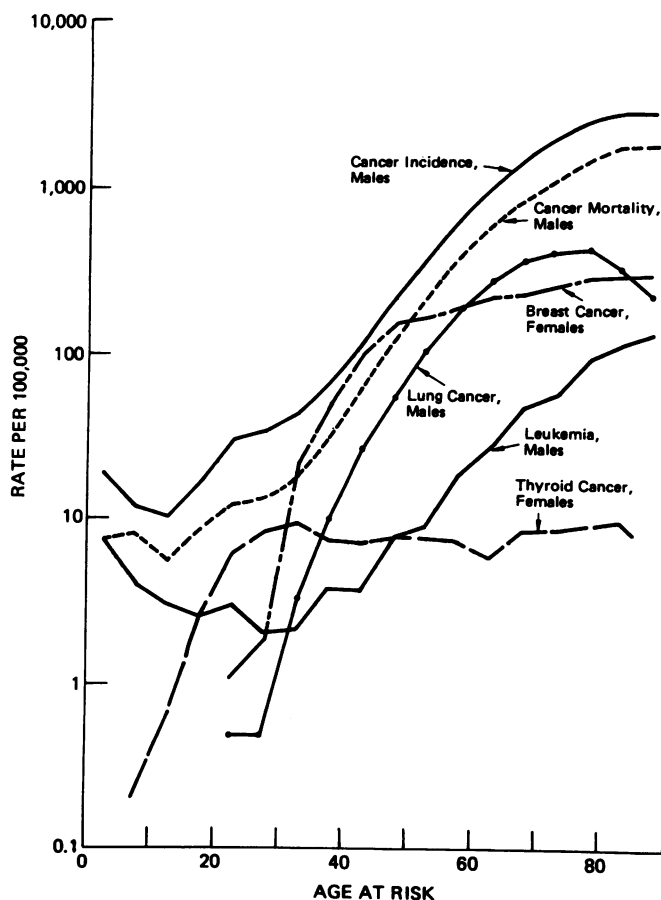
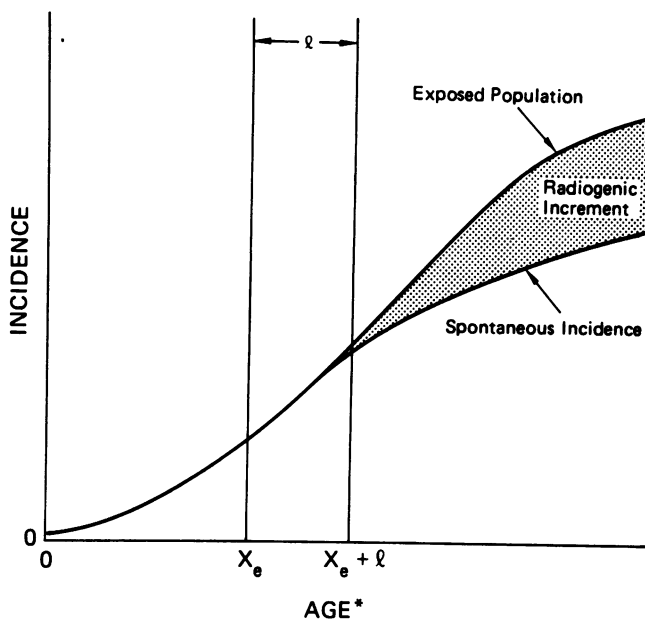


Figure 2.—Age specific cancer mortality rates (from National Academy of Sciences').



* X_e is age at exposure, l is the minimal latent period.

Figure 3.—Radiation cancer incidence. Following exposure (X_e) a minimal latent period (l) precedes the radiogenic cancer increment added to the spontaneous incidence in the exposed population (from National Academy of Sciences').

that our calculations of additional risk from radiation exposure are built.

The risk (Figure 3) associated with each incremental increase in radiation dose can be described in terms of a time following irradiation in which no medical evidence is seen, and is frequently referred to as

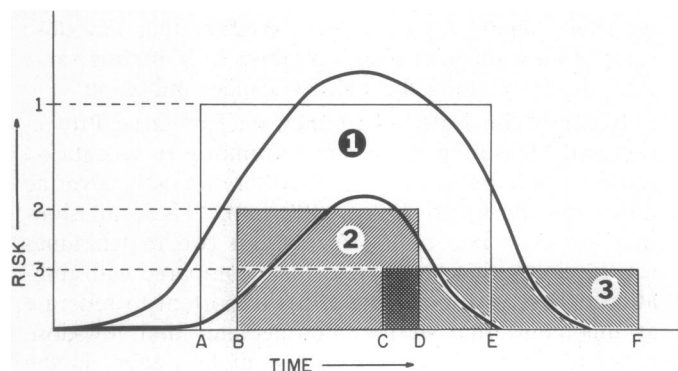
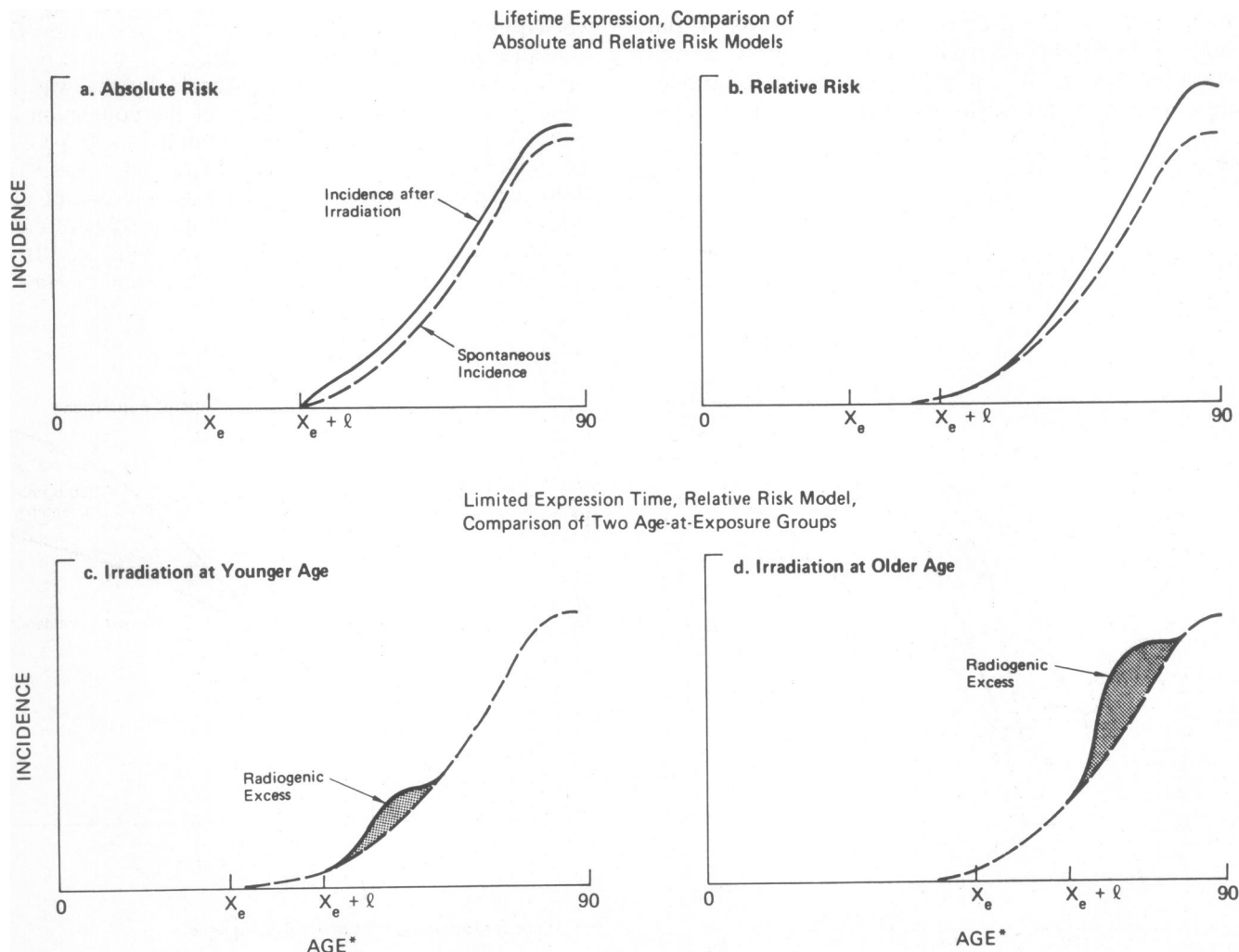


Figure 4.—Latency and sensitivity. These three hypothetical organs each exhibit unique latencies, magnitude of risks and duration of "risk plateau." The data in boxes 1 and 2 are derived from the incidence rate curves shown.

the latent period.⁴ For most solid tumors this is of the order of 5 to 15 years. For leukemia, it is much less, probably two to five years. In a population of exposed persons, this latent period will be followed by an increase in risk. This plateau or height is proportionally related to the intrinsic radiosensitivity of the tissue in question, and whose duration of risk may be short in the case of leukemia, or quite long in the case of solid tumors. Three hypothetical organs and their radiation risks are shown in Figure 4, which is constructed to illustrate this point.

The models utilized to quantify radiation cancer risk are generally summarized in terms of either an absolute or relative risk model for young or old persons. In Figure 5 the National Academy of Sciences recent BEIR (Committee on Biological Effects of Ionizing Radiation) report illustrates the differences between these models.⁴ The relative model assumes a radiogenic cancer risk in proportion to the natural prevalence of the disease in that organ for the given age, whereas an absolute model implies a fixed increment of risk for doses that is independent of age or the "normal" risk for cancer in the organ.



* X_e is age at exposure, ℓ is the minimal latent period.

Figure 5.—Several models of radiation risk (from National Academy of Sciences⁴).

The most conservative approach to radiation risk assessment is to assume a no-threshold linear model, a straight line whose slope predicts that risk increases as dose increases, the assumption here being that each and every incremental increase in dose is associated with corresponding equal increment in risk. Recent radiobiologic studies, particularly those in animals and cells in culture, suggest that the linear model overstates radiation risk at low levels.^{1,7,13,14} In Figure 6, I have attempted to summarize this phenomenon. The small low-dose box containing a question mark is the region of primary concern to occupational medicine and population regulatory interests in that the extrapolation to it from the high-dose data base down to the exceedingly low dose depends upon the model used. The linear model will give a higher predicted risk than the sigmoid model or the dose squared or quadratic model. High-dose rate radiation in particular, and especially that from high-LET emitters such as alpha particles, generally follows a more linear model than does the more conventional low-LET radiation.

Genetic effects of radiation have been known for more than 50 years stemming from the pioneering research on effects of ionizing radiation on *Drosophila*.¹ One very significant finding associated with the early studies was the linearity of the response for the particular endpoints measured. Linearity, however, may not be the case for the mammalian genetic system. Much, if not almost all, of our information regarding the genetic consequences of ionizing radiation stems from important research at Oak Ridge on mice, the "Megamouse Study," in which approximately a three-fold dose rate effectiveness factor was noted for effects in the alleles tested in mice when dose rates were low relative to the yield at the same total dose delivered at higher rates.¹ Ongoing studies of the survivors of the bombings at Hiroshima and Nagasaki have not found genetic effects in the offspring, although much of our knowledge about radiation-induced cancer in humans has been derived from the follow-up of those irradiated populations.⁴

Effects of Radiation on the Embryo and Fetus

If, as Bergonie and Tribondeau predicted, primitive rapidly dividing cells are the most sensitive, one would expect the developing mammal in utero to be particularly vulnerable to ionizing radiation. Much of our observation on this subject derives from studies in rodents in which it was observed that LD₅₀ doses delivered to the embryo invariably resulted in lethality, whereas the same radiation dose delivered to the fetus would induce a spectrum of deformities and anomalies most likely associated with the anlage undergoing differentiation at the time of radiation. It is not known whether embryo death and abortion occurred significantly in pregnant women after the Hiroshima and Nagasaki bombings, but studies on the persons who were at fetal age at time of the bombing have shown only a possibly slight decrease in cranial circumference at birth for those irradiated in the third trimester.¹ The risk for fetal injury based on the mouse model is sufficiently sensitive so as

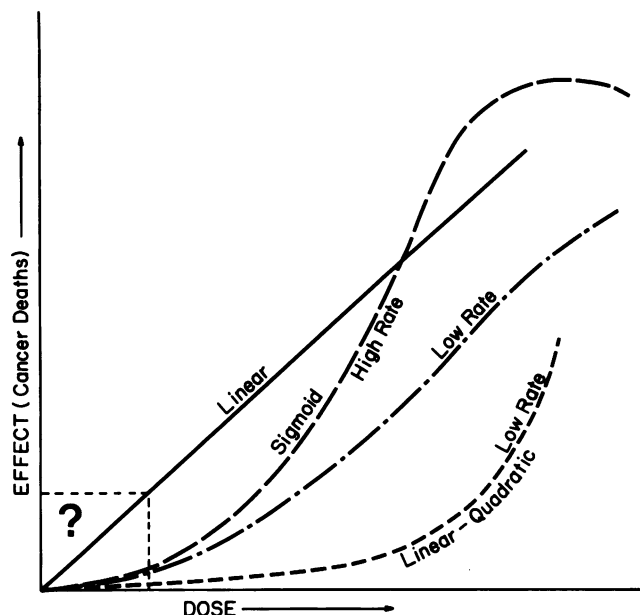


Figure 6.—Radiation risk models. Data fit to the higher dose domain can greatly influence the size of the effect (or risk) prediction in the very low dose range (shown by ?).

to suggest that a fetus receiving in excess of about 10 rem has a high likelihood of being born with a serious defect.⁴ In some instances, abortion is recommended following doses in excess of 10 rem. Women during their fertile years therefore have the added consideration of potential embryonic or fetal exposure associated with radiation-related occupations.

Occupational Exposures

The earliest occupational exposures were among the pioneers in radiology—Röntgen, Becquerel, Curie and the like—who at the time of their remarkable discoveries had no knowledge of the potential health implications of abusing this interesting but difficult to detect form of energy. The early literature is replete with descriptions of lost limbs, blindness, skin lesions and induction of neoplasia. Cancer in early radiologists was particularly common, although it is reassuring to note that the radiology specialty today enjoys about the same cancer mortality statistics as do other medical specialties that do not deal with ionizing radiation.⁴

Early in the century, clock and watch dials were luminized by manually applying to their faces a radium-containing paint.¹⁴ This application with small brushes, usually done by young women, frequently involved "tipping" the brushes with the lips to make the numerals clear and precise. The brush tipping by mouth resulted in the ingestion of small quantities of radium-containing paint. The radium, an alpha-emitting long-lived divalent bone-seeking cationic radionuclide, induced skeletal lesions, including bone cancer and pathologic fractures. Because of its long radioactive and biologic half-life, investigators were provided the opportunity for retrospective dosimetric estimations in people. When the hazards of radium poisoning were recognized and tipping of brushes was prohibited, those

subsequently in that industry have today shown none of these rare lesions. Follow-up and measurement of hundreds of survivors of this industry, as well as some unusual medical detective work, have developed an estimation of risk for this high-LET alpha-emitting radionuclide that strangely appeared not to be linear with cumulative radiation dose, and may even indicate that at low-dose rates the risk, as in low-LET radiation, is curvilinear or that osteoid tissue has some different sort of response.¹⁵

More recently, epidemiological studies of workers at the atomic plants at Hanford, Washington, and those at Portsmouth Naval Shipyard in New Hampshire who were involved in the construction and maintenance of nuclear submarines, and of civilians and military participants in above-ground weapons tests are being closely studied to determine if evidence exists to suggest an increased cancer risk from the work environment. Some preliminary reports have been issued but the final assessment remains to be completed.¹⁶ In each of these groups the major obstacle to the epidemiologic follow-up is the fact that a relatively small number of persons are involved. The radiation risk at low levels is low and in most cases ill defined, and the appropriate control cohort frequently has to contend with problems such as the "healthy worker" syndrome, in which the exposed persons appear to outlive their unexposed comparison group. The range of radiation doses involved and the range of cancer risks implied do not at this time indicate that our present understanding and quantification of radiation risks are grossly misplaced.⁴

Radiation Risk and Regulations

Values from the available literature on radiation risk assessment suggest that a central estimate for fatal cancer in an exposed individual is about 1×10^{-4} per absorbed rem.^{1,4} This is another way of saying that if a million persons were each to receive a rem, approximately 100 additional cancer cases might be added to the 170,000 that would normally be expected in a life-table analysis of such a population size. Risk estimate numbers that ranged widely earlier, now appear to be converging and approximate a value not higher than 200 and probably less than 50. The higher values depend upon the use of a relative risk model. The lower values assume constant radiation sensitivity throughout lifespan and are based on an absolute model. Both the National Academy of Sciences⁴ and the United Nations¹ have published numbers that are quite similar. The same data base is used of course by both groups and in some instances the same experts are doing the analysis.

The case for genetic risks is not quite so clear in view of the absence of confirmatory evidence in humans. However, the best current estimate is that 100 rem delivered to a population would probably double the incidence of genetic defects above that which is normally expected. Another way of summarizing this value is to estimate it in terms of about 60 serious genetic defects per million live births per rem. About

a fourth of these would appear in the very first generation.⁴

Radiation regulations over the years have consistently declined from numbers that initially were stated in terms of radiation exposure limits per day to our current standards in this country as posted in the code of Federal Regulations 10 CFR 20 that workers shall not receive in excess of 5 rem per year, or 3 rem per quarter (sometimes interpreted as 12 rem per year). Somewhat larger annual doses are permitted for the limbs. Regulations also state that the general population shall not receive in excess of a tenth of the occupational level.³ The reasoning here is that those people working with radiation are generally involved in a monitored environment in which exposures are known. This may not be true in the general population.

More recently, radiation protection philosophy has introduced the concept of ALARA, "as low as reasonably achievable." Whatever the dose limit may be in a stated regulation, employers are now required to show that the ionizing radiation exposure potential to workers is to be maintained at levels that are as low as reasonably achievable. This introduces the concept of a collective dose risk problem. For example, if an industrial process entailed a significant radiation exposure potential, is it better to have that radiation absorbed in relatively larger doses by few persons, or to spread the same radiation dose over many people, each receiving smaller doses? In the spirit of ALARA, the latter would be preferable. However, when the same job or work is spread out over a greater number of persons it is not likely that a commensurate increase in efficiency will occur and in general experience has shown that the *total* amount of radiation absorbed by the worker population assigned to a task increases as the average or unit or individual radiation dose decreases. The risk one would calculate, therefore, is highly dependent on whether one uses a curvilinear or a linear model for this low-dose domain. A linear model would say that spreading the dose out increases the risk. The curvilinear model says the opposite. Time will show which, if either, is correct.

Radiation Issues in Society

The radiation issue is probably one of the most widely discussed in the professional and lay press. Some of the issues discussed above that have yet to be resolved involve the precise quantification of the dose-response relationship. Is it linear or is it something else? If so, how can we prove it? Buried in this particular issue is another issue regarding the purported supersensitivity of certain small subsets of the population, for example, those who may be considered to be at high risk for cancer or genetic problems, or some other medical condition. For these persons should the standards be lower?

A second issue of considerable importance is the potential synergism between the physical risk associated with ionizing radiation and the potential environmental cancer risks from chemicals. If the same organs are at risk will small absorbed doses of both act synergistic-

ally, antagonistically or in an unrelated fashion? The literature on this particular point supports a variety of hypotheses, but in no way is the answer clear at this time.

An issue that goes beyond radiation biology relates to the problem of fertile women in the workplace. If the fetus and embryo are uniquely sensitive, then the standards relating to the fetus may be imposed on the woman; this provides the potential for discriminatory actions in order to protect the unborn.

Another issue relates to the quality of radiation. Over the past few decades, it has been conventional in the formulation of regulations and in the discussion of qualities of radiation to ascribe a constant factor, called a quality factor or a relative biologic effectiveness factor, to relate, for example, one type of LET radiation to another type of LET radiation. That the endpoints differ for different doses and rates and that the nature of the response curves for the two compared radiations appear not to be linear imply that a simple ratio is no longer applicable for risk estimation purposes. Instead a range of comparisons may be more appropriate.⁸ An extreme example is in the case of comparison of high-LET alpha particles to low-LET x-rays at doses in which the x-ray effect approaches zero. When the denominator approaches zero the ratio approaches infinity; the inference would be that the high-LET radiation has some unique potency at low levels rather than that the comparison had almost no impact.

Lastly, what may appear to be more of a political issue is my observation that as information has been gleaned about the quantification of radiation risk, society appears less inclined to accept these values. This is not to imply that ignorance is bliss and that knowledge is a liability, but at times it is difficult to reconcile with the facts the difference between not having done

the risk estimation and not liking what the risk estimation says once it has been done. In my opinion, radiation risk estimation represents a prototype for occupational hazards from other exposures. The scientific/societal interaction with these radiation risk estimates will likely set the precedent for many other agents whose potential occupational health implications we are just now learning to question.

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